Modern Concepts of Cardiovascular Disease

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CARDIAC ARREST DURING SURGERY*

A significant advance in recent years has been the treatment and prevention of cardiac arrest occurring during surgery. Cardiac arrest does not happen only in the operating room but may occur as a result of drug reactions, electrocution, and other unfortunate accidents. The purpose of this discussion, however, is to summarize briefly the present state of knowledge regarding the best methods of treatment and prevention of surgical cardiac arrest. It must be emphasized at this point that methods of treatment (emergency thoracotomy and cardiac massage) appropriate for surgical cardiac arrest are not to be applied to persons dying from cardiac disease, renal disease, metastatic cancer, cerebrovascular accidents, and similar conditions.

DEFINITION

By cardiac arrest is meant the sudden cessation in the ability of the heart to maintain the movement of blood through the circulatory system. This may be due to complete cardiac standstill (the commonest occurrence), ventricular fibrillation, or slow ineffectual contractions of the heart. Only minutes separate life from death in this situation, and an irresolute hand will not be successful here. The cells of the central nervous system are highly susceptible to anoxia. They are destroyed rapidly and cannot be replaced or rejuvenated. If the circulation is lacking for longer than three or four minutes, it is highly unlikely that complete recovery can occur. There may be survival but in a decerebrate state.[1] After seven or eight minutes of cerebral anoxia, death is inevitable. Thus, because so little time is available one should be prepared, both psychologically and technically, to remedy this difficult situation. If circulation can be effectively restored within one or two minutes, complete recovery is possible[2] but is proportionately diminished if hypoxia or anoxia continues beyond this time.

DIAGNOSIS

Obviously the length of time required to make the diagnosis of cardiac arrest is of critical importance. The diagnosis during a surgical procedure is usually made by the anesthetist unless the chest is open or large vessles are exposed. The outcome, therefore, depends in a large degree on close cooperation between the surgeon and the anesthetist. The only reliable signs of cardiac arrest are absence of the blood pressure and pulse. One must not depend upon cessation of respiration since breathing may continue for a few moments after cardiac action has ceased. As soon as the anesthetist reports that a blood pressure is not obtainable and a pulse cannot be felt, the surgeon should suspect that cardiac arrest has occurred. In the case where the heart and great vessels are neither visible nor palpable, Johnson and Kirby[3] have taken the position that a thoracotomy becomes both a diagnostic and therapeutic procedure and should be promptly performed. For success to attend this procedure a means of adequate artificial respiration must be immediately available. Sterility is desirable but should not delay thoracotomy. Sterile technique should be initiated, however, as soon as possible.

Certain warning signs may occur prior to cardiac arrest, and careful observation will frequently give some indication of an impending catastrophe. Respiration may increase both in rate and depth. Cyanosis may be present. With the early onset of hypoxia the blood pressure may rise and later fall to a lower level than it was initially. Cardiac arrest may occur forthwith. In addition frequent ventricular premature beats, excessive bradycardia, or tachycardia may precede the cessation of the heart beat.[2] These findings, of course, are not necessarily seen prior to arrest.

When blood pressure, pulse, and respirations become unobtainable, one should not waste time by first taking an electrocardiogram, looking in the eyegrounds, or carrying out conservative attempts to restore cardiac function. One should listen briefly to the chest if a stethoscope is immediately available. It is true that immediate opening of the chest under these circumstances may result in unnecessary thoracotomy for other disastrous complications resembling cardiac arrest. This procedure has been carried out when pulmonary embolism and coronary occlusion developed on the operating table. Such relatively infrequent errors will have to be accepted, however, if patients with surgical arrest are to be saved.[1]

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ETIOLOGY

Although sometimes unaccountable, cardiac arrest associated with surgical procedures can usually be ascribed to one or more of the following:

(1) *Direct manipulation of the heart* or other intrathoracic organs.

(2) *Hypoxia.* This is the most widely incriminated of all factors causing cardiac arrest. Cardiac muscle is more senstitive to hypoxia than skeletal muscle[4]. Many obvious causes for hypoxia exist, such as laryngo-tracheal obstruction, respiratory depression by excessive anesthesia, pulmonary disease, aspiration, and cardiac disease. Anemia and shock occurring in a surgical procedure may also lead to acute hypoxia and cardiac arrest.

(3) *Excessive anesthesia* or sensitivity to the anesthetic agent.

(4) *Vagal reflexes.* The exact role of the vagal reflexes in producing cardiac arrest has not been settled. During electrical stimulation of one vagus nerve proximal to the heart in the human being, no particular arrhythmias were seen if oxygenation was adequate.[5] In laboratory animals cardiac arrest does not seem to occur by vagal stimulation in the absence of hypercapnea or hypoxia.[6] In the human being during endotracheal intubation there are reports of electrocardiographic changes which were thought to be related directly to hypoxia as measured by the oximeter. Vagal stimulation was not considered to be an important cause of arrhythmias in the presence of satisfactory oxygenation.[5]

(5) *The presence of previous cardiac disease.* Coronary artery disease with areas of hyperirritable ischemic myocardium predisposes to the development of cardiac arrhythmias during surgery and particularly to the onset of ventricular fibrillation.

(6) *Other factors.* Malnutrition, fright prior to operation, carbon dioxide accumulation during surgery and its rapid reduction postoperatively have been suggested as causes of postanesthesia cardiac arrest.[7]

PROPHYLAXIS

The surgeon should not demand that the patient be carried too deeply under anesthesia. The anesthetist must be astute in his choice of anesthesia and must guard against using excessive amounts. A proper airway must be maintained with avoidance of hypoxia and hypercapnea. The premonitory signs of an impending disaster must be watched for constantly.[3] Anemia and malnutrition should be corrected prior to the surgical procedure. Blood lost during the procedure should be replaced immediately. Epinephrine should be used only rarely, if at all, during the administration of cyclopropane. If the myocardium shows evidence of excessive irritability prior to surgery, quinidine or Pronestyl® are probably worthwhile given one hour preoperatively. Where surgery is mandatory in patients with known cardiac disease, time should be allowed preoperatively to effect as much improvement as posible in the cardiac status. For example, a delay of only a few hours, while a decompensated patient is digitalized, may mean the difference between survival and death. Any metabolic disturbances such as dehydration and acidosis should be corrected if possible. Seldom does one have to be in such a hurry that a few hours cannot be allowed for attempted correction of these serious defects.

In cardiac surgery the condition which is most likely to lead to a disaster is ventricular fibrillation. There is little evidence that preoperative administration of quinidine or Pronestyl® is of benefit in preventing this condition. The most important prophylactic measure is not drug therapy but rather adequate oxygenation of the myocardium.[3]

The maintenance of a proper mental attitude in the patient just before surgery requires reassurance and adequate preoperative medication.

Sudden changes in position of the patient during anesthesia and surgery should be avoided.[2]

INCIDENCE

Several cases of cardiac arrest occurring during surgery may probably be expected in any large hospital each year.[8] Arrest may occur at any age, with any type of anesthesia, and in good or poor risk patients.[2]

TREATMENT

If a well thought out program of action for the handling of cardiac arrest is planned in advance, the surgeon and anesthetist will be able to act resolutely in the face of this catastrophe and can institute immediate resuscitative measures. Techniques were outlined by Beck in 1937 for the control of the heart beat by the surgeon at the operating table.[9] A clear understanding of the necessary treatment can be obtained if one keeps in mind the objectives emphasized by Beck,[10] namely, restoration of the oxygen system and restoration of the heart beat.

Restoration of the Oxygen System

The type of arrest in the initial phase of treatment is unimportant. The object in all forms is to re-establish an effective circulation with the least possible delay. The most effective way to do this is cardiac massage with the hands directly on the heart. Cardiac arrest indicates the need for immediate thoracotomy. Johnson and Kirby have demonstrated that direct massage through a transthoracic incision produces a five times greater cardiac output than massage carried out subdiaphragmatically and two times more cardiac output than massage carried out through a transdiaphragmatic incision. Many surgeons feel it is also advantageous to open the pericardium when massage is to be undertaken. A detailed discussion of the methods of cardiac massage is

beyond the scope of this paper. The reader is referred to the articles in the bibliography for a more complete description of these techniques.

The proper rate of cardiac massage is in some dispute. Regardless of the rate used, which varies from 25 to 80 or more per minute[5, 11], practice in massaging is probably the most important factor in getting good results. The effectiveness of the massage can be judged by the presence of a palpable pulse, bleeding from the wound, and a good color.[3] If cyanosis persists, the airway, oxygen tank, and soda lime cannister should be checked. The Trendelenburg position is favored during massage.[2]

The immediate initiation of artificial respiration is equally as important as cardiac massage. This may be mouth-to-mouth initially until the proper equipment can be obtained. The best equipment is an anesthesia machine which can deliver 100% oxygen through a face mask. An endotracheal tube should be inserted, but, before this is attempted, the patient should be well oxygenated by the bag and mask. The bag should be emptied repeatedly and refilled with oxygen to wash out all inhalation anesthetic agents. A mechanical respirator, if available, will be of great value.

Restoration of the Heart Beat

(a) *Cardiac Standstill.* In many cases of cardiac arrest due to asystole, massage alone will in a few moments restore an effective spontaneous heart beat. If spontaneous heart beat does not result, a dilute adrenalin solution (1:10,000) can be injected into the right chambers of the heart and repeated several times if necessary. If this procedure is ineffective, 10% calcium chloride can be injected.[12]

(b) *Ventricular Fibrillation.* This condition is fortunately less common than asystole. When fibrillation is present, the mainstay of immediate treatment is cardiac massage to maintain an effective peripheral and coronary blood flow and to overcome the cardiac dilatation and myocardial cyanosis which is usually present. In the meantime preparations should be in progress to defibrillate the heart, defibrillation being absolutely necessary before spontaneous effective cardiac contractions will resume. Little success will attend attempts to defibrillate a dilated and cyanotic heart. Such efforts should be delayed until muscle tone and color have been restored by massage.[9]

The defibrillatory apparatus is equipped with electrodes capable of delivering an electric current directly to the heart. Ideally this instrument should also contain a device for accurately timing the duration of the electrical shock, an isolation transformer for reducing the risk of shocking the operator, and, if feasible, a device for varying the amount of voltage.

The use of 110 to 135 volts should be routine. Occasionally greater voltage may be tried in large hearts that do not respond to the lower voltage.[3]

A series of short shocks is probably more satisfactory than one long shock, and these should be given until defibrillation has occurred. A good plan is to deliver three to five 110 volt alternating-current shocks, each of 0.1 second duration.[13] The interval between each shock should be about 0.5 to 1.0 second.

The defibrillating electrodes should be as large as ventricular size will permit. The heart should be firmly compressed between the electrodes during the flow of current.[14] This reduces resistance and aids in more complete defibrillation.

The surgeon must take care not to shock himself. He can best prevent shock by wearing two pairs of gloves, by having insulated electrode handles, by having an isolation transformer, if possible, and by not touching the table. The anesthetist should not touch either the table or the patient during the period in which current is flowing. If the entire body of the patient does not jerk at the application of the electroshock, the current is probably not strong enough.[3]

An electrocardiograph should be used since this instrument provides the best way of determining when defibrillation has occurred and the nature of the subsequent rhythm. It must be disconnected during the use of electrical shock.

If electroshock alone is unsuccessful in securing defibrillation, the surface of the heart can be sprayed with 1% procaine, or 3 to 5 cc. of 1% procaine can be injected into the right ventricle. Electroshock and massage can then be repeated. The procaine drugs, however, are not without depressant effects on arterial pressure, respiration, and the myocardium.[14] They should not be used unless really necessary.

When electroshock is effective, cardiac standstill usually results, and, with continued massage, the ventricles should resume effective contractions. Occasionally the heart after defibrillation remains flabby and contracts ineffectually. In this situation, small amounts of dilute epinephrine solution can be injected into the right atrium or right ventricle coincident with massage. If epinephrine is unsuccessful, 2 to 4 cc. of 10% calcium chloride solution may be injected into the left ventricular cavity and massage continued. These procedures may be repeated after several minutes if necessary.[12] Since recovery of the heart beat has been reported after an hour or more of fibrillation,[15] perseverance in treatment is important.

PROGNOSIS

Prognosis for recovery from arrest[2] depends on the following:

(1) The length of time elapsing before massage is begun.

(2) The mechanism of arrest, fibrillation generally carrying the poorer prognosis.

(3) The degree of hypoxia present and the status of the glycogen reserves.

(4) The presence or absence of pre-existing heart disease.

(5) The presence of noxious drugs.

(6) The degree of diastolic filling and coronary blood flow that can be maintained by massage.

CONCLUSION

Prompt and vigorous treatment of cardiac arrest during surgery has produced an increasing number of recoveries from this catastrophe. Treatment failure, however, remains high, especially in ventricular fibrillation. A further improvement in mortality figures should be possible with a wider understanding of the mechanisms involved and an appreciation of the opportunities that exist for therapy.

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The opinions and conclusions expressed herein are those of the author and do not necessarily represent the official views of the Scientific Council of the American Heart Association.

PAPERS FOR THE SCIENTIFIC SESSIONS

All those wishing to present papers at the 28th Scientific Sessions of the American Heart Association must submit abstracts for review by the Program Committee. Abstracts shall not be more than *300 words* and must be submitted in duplicate before *July 1, 1955*, to the Medical Director, American Heart Association, 44 East 23rd Street, New York 10, New York. Abstracts should contain in summary form the results obtained and conclusions reached — *not* a statement that these will be presented at the meeting.

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